A STUDY OF CLINICAL PRESENTATION AND HISTOPATHOLOGY OF CHILDHOOD LEPROSY

Dissertation Submitted to

THE TAMIL NADU DR. M.G.R. MEDICAL UNIVERSITY

in partial fulfillment of the regulations

for the award of the degree of

M.D. (Dermatology, Venereology and Leprology)
BRANCH – XII A

MADRAS MEDICAL COLLEGE
THE TAMIL NADU DR. M.G.R. MEDICAL UNIVERSITY
CHENNAI, INDIA.

MARCH 2007

CERTIFICATE

Certified that this dissertation entitled **"A STUDY OF CLINICAL PRESENTATION AND HISTOPATHOLOGY OF CHILDHOOD LEPROSY"** is a bonafide work done by **Dr. S.MURUGAN**, Post graduate student of the Department of Dermatology and Leprology and Institute of Venereology, Madras Medical College, Chennai- 3, during the academic year 2004 – 2007. This work has not previously formed the basis for the award of any degree or diploma.

Prof. Dr. B. PARVEEN, M.D., D.D.,
Professor and Head of the Department,
Department of Dermatology and Leprology,
Madras Medical College,
Chennai- 3.

Prof. Dr .KALAVATHI PONNIRAIVAN, B.Sc., M.D.,
DEAN, Madras Medical College,
Chennai- 3.

DECLARATION

I, **Dr. S.MURUGAN,** solemnly declare that dissertation titled, "**A STUDY OF CLINICAL PRESENTATION AND HISTOPATHOLOGY OF CHILDHOOD LEPROSY"** is a bonafide work done by me at Madras Medical College during 2004-2007 under the guidance and supervision of **Prof. Dr. B. PARVEEN, M.D.,D.D.,** Professor and Head, Department of Dermatology, Madras Medical College, Chennai-600 003.

The dissertation is submitted to The Tamilnadu, Dr. M.G.R. Medical University, towards partial fulfillment of requirement for the award of **M.D. Degree in Dermatology, Venereology and Leprology (BRANCH – XII A).**

Place : Chennai.

Date :

(Dr. S.MURUGAN)

SPECIAL ACKNOWLEDGMENT

My sincere thanks to

Prof. Dr .KALAVATHI PONNIRAIVAN, B.Sc., M.D.,
DEAN,
Madras Medical College

for allowing me to do this
Dissertation and utilize the institutional facilities.

ACKNOWLEDGEMENT

I am gratefully indebted to **Prof. Dr. B. Parveen M.D., D.D.,** Professor and Head, Department of Dermatology and Leprology for her invaluable guidance, motivation and help though out the study. I would like to express my sincere and heartfelt gratitude to **Prof. Dr. V.S. Dorairaj, M.D., D.V.,** Director in charge, Institute of Venereology.

I wish to thank Dr.N.Gomathy, M.D., D.D., former Professor, Department of Dermatology and Dr. N. Usman M.D., D.V., Ph.D., former Director, Institute of Venereology for their constant support and motivation.

I am very grateful to Dr. S. Jayakumar M.D., D.D., Additional Professor, Department of Dermatology for his invaluable guidance and help. I sincerely thank Dr. C. Janaki M.D., D.D., Reader of Dermatology (Mycology) for her priceless support.

I express my earnest gratefulness to Dr. D. Prabavathy M.D., D.D., Professor and Head of Department of Occupational Dermatology and Contact Dermatitis for her constant motivation and guidance. I thank Dr. V. Somasundaram M.D., D.D., Additional Professor, Department of Occupational Dermatology and Contact Dermatitis for his benevolent help and support.

I express my sincere gratitude to Dr. K. Rathinavelu M.D., D.D., Professor of Leprosy and Dr. R. Arunadevi M.D., D.D., Lecturer/Registrar, Department of Dermatology for their support.

I incline to thank Dr.R.Priyavathani, M.D.,D.D., D.N.B., Dr.V. Anandan M.D.,(Derm),D.C.H.,D.N.B.,(Paed) and Dr.G.K. Tharini M.D., Dr.Vijayanand M.D., Assistant Professors, Department of Dermatology for their kind support and encouragement.

I thank Dr. A. Hameedullah M.D., D.D., Dr. S. Kumaravelu M.D., D.D., Dr. J. Manjula M.D., D.N.B., (Derm) and Dr.Aftab Jameela Wahab M.D., D.D., Assistant Professors, Department of Occupational Dermatology and Contact Dermatitis for their support and help.

My sincere thanks of Dr.S.Mohan, M.D., D.V. former Registrar, Dr.V.Thirunavukkarasu, M.D., D.V., Dr.K.Venkateswaran, M.D., D.V., Dr.P.Elangovan, M.D., D.V., DR.D.Ramachandra Reddy, M.D., D.V., Dr.S.Thilagavathy, M.D., D.V., Dr.P.Mohan, M.D., D.V., S.Arunkumar, M.D.,D.V., and Dr.S.Kalaivani, M.D.,D.V., Assistant Professors, Institute of Venereology for their help and suggestions.

I am also thankful to Dr.K.Manoharan, M.D.,D.D., and Dr. V. Sampath M.D., D.D., for their continuing guidance and support.

I duly acknowledge the paramedical staff and my colleagues for their help and favour.

Last but not least I am profoundly grateful to all patients for their cooperation and participation in the study.

CONTENTS

ABBREVIATIONS

IND	-	Indeterminate
TT	-	Tuberculoid
BT	-	Borderline Tuberculoid
BL	-	Borderline Lepromatous
PNL	-	Pure Neuritic Leprosy
Hypo P	-	Hypopigmented
Ery	-	Erythematous
Cop C	-	Copper coloured
B.UL	-	Both ulnar
L.UL	-	Left ulnar
R.UL	-	Right ulnar
R.MN	-	Right median
L.MN	-	Left median
B.MN	-	Both median
B.LP	-	Both lateral popliteal
L.LP	-	Left lateral popliteal
R.LP	-	Right lateral popliteal
L.PT	-	Left posterior tibial
R.PT	-	Right posterior tibial
B.PT	-	Both posterior tibial
R.GA	-	Right greater auricular
L.GA	-	Left greater auricular
B.GA	-	Both greater auricular
B.RC	-	Both radial cutaneous
L.RC	-	Left radial cutaneous
R.RC	-	Right radial cutaneous
R.SU	-	Right sural
L.SU	-	Left sural
L.SC	-	Left supra clavicular
L.SO	-	Left supra orbital
R.SO	-	Right supra orbital
L.IC	-	Left intermediate cutaneous nerve of thigh
L.LC	-	Left lateral cutaneous nerve of thigh
L.MC	-	Left medial cutaneous nerve of thigh
HP	-	Histopathology
SSS	-	Slit skin smear

INTRODUCTION

Leprosy is one of the oldest disease of mankind. Eventhough tremendous progress has been made in the field of leprosy elimination, it still continues to be an endemic problem in certain parts of India. India represents close to 76% of the global burden.

Leprosy is a well documented entity in children. The overall prevalence of leprosy in India has declined from 5.27 / 10000 in the year 2000 to 1.34 / 10000 in the year 2005, still it constitutes a sizable health problem in the pediatric age group with an incidence of 13.3 % in children between the age group 0 -14years. Children between 0 - 14 years age group constitute approximately 30 % of the population.

The worldwide prevalence of leprosy has decreased dramatically, since the inception of elimination plan, but the disease deduction rate has remained almost constant over the past ten years, with a high rate (17 %) of infection in children.

Leprosy in children has epidemiological significance and can be considered as an index of the prevalence of the disease in the population. A high child rate may indicate continuing spread of the disease in the community. A case detected among children also provides an opportunity to detect the index case usually with in the family.

Leprosy in children forms an important link in the study of the natural evolution of the disease. The spectrum of the disease is usually incomplete in children. It could be said that leprosy in children reflects to some extent practically all aspects of the disease in adults, with additional features of its own.

Leprosy in children is common, but rare under 2 years of age. It is often unrecognized and 75% of cases regress spontaneously without treatment. Prevalence rate increases regularly in the age group 0-4, 5-9 and 10-14 years. The youngest age reported for occurrence of leprosy is 3 weeks in Martinique. The youngest case of tuberculoid leprosy confirmed by histopathology was in an infant of 2.5months old. Leprosy in children is equally prevalent in both sexes.

Children are susceptible to leprosy, as the immune system is not fully developed. In children it presents predominantly as paucibacillary indeterminate, tuberculoid, borderline tuberculoid and occasionally borderline borderline, borderline tuberculoid being the most commonest. Borderline lepromatous and lepromatous leprosy are rare. This appears to be a paradoxical situation in children and runs counter to the concept that immune responses are either negligible or poorly developed in children**.**

Childhood leprosy usually responds rapidly to treatment. Reactions and relapses are not uncommon in children. Ocular leprosy, deformities, infectivity, poor tolerance to antileprosy drugs, and special variants like histoid leprosy are rare in children.

REVIEW OF LITERATURE

Definition

Leprosy is a chronic disease caused by Mycobacterium leprae, infectious in some cases, and affecting the peripheral nervous system, the skin, and certain other issues [1]

History of Leprosy

Leprosy is a very ancient disease. Its origin and early spread is not definitely known. Leprosy is prevalent for many centuries in India, Africa and China. Some believe it first originated in Asia and others in Africa in Egypt.

In India leprosy was first described in the Susruth Samhita in 600 BC and treatment with chaulmoogra oil was known at that time [2]. Leprosy in India has been mentioned in the Vedic writing as kusht around 1400BC.

The disease was probably carried from India to Europe in the 4th century BC by returning soldiers and camp followers from the Greek wars of conquest in Asia, led by Alexander the Great, and the earliest description of a disease which was unmistakably leprosy was by Aretaeus, in Greece, about 150 A.D as elephantiasis. [3]

Noble families founded Leprosoria, hospitals for leprosy patients in twelfth and thirteenth centuries. [4] Leprosy patients were legally considered as dead during that period.

Moller Christensen's (1961) work revealed that 80 % of the skeleton excavated at Naestved, Denmark, the burial ground of Lazar hospital which existed between 1250 and 1550 A.D. showed pathognomonic changes of leprosy. [4] The Naestved study revealed that the sex incidence was equal and the majority of the leprosy cases had onset in childhood. [4]

Carl William Boeck (1808 – 75) and Daniel Cornelius Danielssen (1818-94) are two of the most renowned leprosy experts of the nineteenth century, believed that leprosy was a congenital disease and not an infectious one.

Gerhard Henrick Armauer Hansen (1841-1912) Danielssen's son-in-law discovered the causative organism of leprosy Mycobacterium leprae in 1873. [5]

EPIDEMIOLOGY

Geographical distribution

Although the worldwide prevalence of leprosy is less than one per 10000, it is still a public health problem in 15 countries including India, Brazil, Myanmar and Nepal. [6].

In India the prevalence rate (PR) has reduced from 5.27/10000 in March 2000 (5,18,000 cases on record) to a PR of 1.34/10000 in March 2005 (1,49,000 cases on record). [7] India contributes about 64% of world's registered cases. [6] Of the 35 states / union territories in the country 24 have reached goal of elimination 7 states – Bihar, Chattishgarh, Jharkhand, Maharastra, Orissa, Uthar Pradesh, Andhra Pradesh, West Bengal and four

union territories Chandigarh, Dadra Nager Haveli, Delhi and Goa did not reach the goal. [8] Incidence in Tamilnadu is 0.95/10000.

Predisposing factors [9,10]

Over crowding

Poor sanitation

Mal nutrition

Factors in transmission of leprosy [9]

1. Source of infection
2. Modes of transmission
3. Susceptibility of host

Source of infection

The only source of infection is a leprosy patient. All patients are not infective but only those patients who are capable of discharging bacilli from their body are known as infectious or open cases belong to the lepromatous pole. The patients who are unable to shed bacilli are known as non-infectious or closed cases, belong to the tuberculoid pole, most cases among children belong to this group.

When the primary case was lepromatous the incidence rate was 6.24 per 1000 PYR (persons years of risk) and if it was non-lepromatous it was 1.6 per 1000 PYR [11,12] Source of contact may be intrafamilial or extrafamilial.

Intrafamilial contact: The number of cases among contacts was more in the paediatric (0-14 years) age group, 62.5 %. [13,1,11] The maximum prevalence of 45.8 % is in the 6 – 14 years age group. The source of majority of cases was the father (57%) followed by the mother (22.5 %), siblings 9.8 % [13,14] The index case was a grand parent in 14% cases. [15]

Extrafamilial contact: The source of infection in many of the school children is extrafamilial because of the presence of high quantum of infection in the community. [9]

The risk of a person developing leprosy is 4 times higher when the leprosy contact is in neighborhood; the risk is increased to nine times if the contact is within immediate household and even higher if the cases are multi-bacillary. [16]

When the type of leprosy in the adult contact was known, it was seen that relatives with multibacillary disease had been having it for years before the children were affected, in contrast, with the paucibacillary disease, the relative and the child were affected within a few months of each other, when considering the long incubation period of the disease, the latter may be due to the acquisition of the infection by both the child and the relative from an unrecognized multibacillary source.[14]

Modes of Transmission

Portal of exit : The two main portal of exit of M.leprae are the skin [17,18] and nasal mucosa, the latter being the most important one. [19] Other portal of exit are breast milk and female genital mucosa. [20]

Portal of entry : Skin [17,18] and the upper respiratory tract are the two main modes of entry. The organism may also enter thro gastrointestinal tract[20] and transplacental transmission may occur as a rare event. [1]

Susceptibility of host

The development of the disease and the spectrum are determined by the degree of specific immunity of the infected person against the leprosy bacilli. [8] Children are more susceptible as the immunity is poorly developed.

Duration of contact

Closeness and duration of contact are of importance in determining the dose of infection, but in highly susceptible person even a casual contact may result in infection. [9] Maximum number of cases acquired the disease in 4-6 years of contact followed by 0- 3 years of contact. [13]

Age

Leprosy is known to occur at all ages from infancy to very old age. The incidence is higher in 10 – 14 years of age and 30 - 60 years of age group. [19] The mean age at onset of leprosy in males was 31.49 years and female 29.43 years [21]

Leprosy in children below 2 years of age is very rare. [22] The youngest age reported for occurrence of leprosy is 3 weeks in Martinique. The youngest age of tuberculoid leprosy confirmed by histopathology is 2.5 months old age. [19]

Prevalence rate in children increases regularly in the age group 0 - 4, 5 – 9 and 10 – 14 years [13,22,23,24,25,26,41].

Incidence of leprosy in children 0 –14 years is 13.3 %, March 2005. [7] Children in this age group constitute approximately 30% of population.

Sex

Leprosy in adults is more prevalent among males than females. In children there is no significant difference in leprosy prevalence between sexes. [22]

Most studies have reported male female ratio that favors males [6,13,25,26,27,28,41]

Incubation period

It may be as short as few months as long as 20 years or more. On an average it is between 2 – 5 years. [9,19]

Vaccination

Prevalence of leprosy is about 2 times more common among non-vaccinated children than BCG vaccinated children. [29]

BCG vaccination provides 50% protection among contact children, varies between 20 –80 % [29,30]

Genetic factors

HLA association

Tuberculoid Leprosy DR3

Lepromatous Leprosy DQ1 [31]

CLASSIFICATION [32]

International classification at Madrid (1953)

1. Lepromatous Type (L)
   - Macular
   - Diffuse
   - Infiltrated
   - Nodular
   - Neuritic, pure (?)
2. Tuberculoid Type(T)
   - Macular (Tm)
   - Minor tuberculoid (Tt)
   - Major tuberculoid (TT)
   - Neuritic, pure (Tn)
3. Indeterminate Group (I)
   - Macular (Im)
   - Neuritic, pure (In)

4. Borderline Group (B)

Infiltrated

(Others?)

Revised Indian Classification (1981)

Tuberculoid

Borderline

Lepromatous

Indeterminate

Pureneuritic

Ridley and Jopling (1962)

Tuberculoid (TT)

Borderline tuberculoid (BT)

Borderline Borderline (BB)

Borderline lepromatous (BL)

Lepromatous Leprosy (LL)

WHO classification (1982)

Paucibacillary

Less than 5 skin lesions

No nerve involvement or one nerve involvement

Bacterial index < 2+

Multibacilliary

More than 5 skin lesions

More than one nerve involvement

Bacterial index ≥ 2+

(Changed in1988 to paucibacillary = bacterial index of 0; multibacillary ≥ 1+, WHO Expert committee on leprosy 1988)

Clinical features

Leprosy may manifest in one of the four ways, cutaneous lesions, neural symptoms, reactional episodes and deformities. In children reactional episodes and deformities are rarely the presenting features. [22]

Cardinal signs

- Anesthetic skin lesions
- Nerve enlargement
- Demonstration of Mycobacterium leprae in slit skin smear

Early signs of the disease

The early signs mainly affect the skin and to a lesser extent the peripheral nerves. [22]

The early signs include [9]

- A patch in the skin which is lighter in color than the surrounding, persist for long, does not irritate or itch, and in which there is partial or total loss of sensation ,temperature, pain and light touch.

- Thickening of skin, red or shiny especially prominent in face and hands.

- Numbness or feeling of pins and needles or crawling of ants or tingling sensation especially in hands and feet or weakness in fine movements of fingers.

- Burns resulting from contact with hot articles, which at the time of contact did not cause pain.

- Appearance of spontaneous blisters and ulcer especially in fingers.

Prodromal symptoms are rarely identified and have no clinical or diagnostic significant.

Skin Lesions

Skin lesions may be single or multiple approximately 90% of the leprosy patients had skin lesion and 79.5 % had skin lesions only. [22]. Single skin lesion is more common followed by 2 to 3 and more than 4 is rare. [22,26,28] The sites for the development of the single lesions of leprosy in children are predominantly seen on the exposed parts [15,22,33,34,35,36,37,38,39,41,42], thighs and buttocks followed by arms, forearms, legs, trunk especially lumbar region, face and neck. [22,15,34,40]

The leprosy skin lesions found in children are not essentially different from those seen in adults, although in children the lesions in majority of case not so well defined, of smaller size and less in number. [22] The main diagnostic sign the loss of superficial sensation within the lesion in the order of temperature, pin prick and light touch is a constant feature of tuberculoid and borderline tuberculoid but not a feature of early borderline borderline,

borderline lepromatous and lepromatous leprosy. In indeterminate leprosy pain and temperature is impaired with exception but light touch is not frequently impaired. [22]

The most common type of leprosy skin lesion found in children is hypo pigmented macules and infiltrated patches. Lichenoid lesions, papulo-nodules and weal like papules are less common. Secondary lesions like ulcers and scars are rare. [22]

Nerve involvement

Leprosy is one of the most important causes of nerve enlargement. Nerve enlargement occurs approximately about 20.4 % and exclusive nerve involvement in 10.6 % in childhood leprosy. [22]

Peripheral nerve commonly enlarged are superficial nerve trunks; ulnar, lateral popliteal and greater auricular are frequently affected in that order. Other nerves like posterior tibial, median, radial, and facial and cutaneous nerves like radial cutaneous, supraclavicular, supraorbital and sural nerve are also felt.[22,42]

Apart from the nerve trunks and the cutaneous nerves there may be enlargement of small superficial nerves supplying the suspicious macule is of great diagnostic significance, more common in tuberculoid patch.

Muscular paralysis with or without contracture is infrequent in children and advanced deformities, bone absorption and loss of fingers or toes are exceptional. [22]

Other signs such as ocular lesions, nose involvement, loss of eyebrows, laryngitis, ear lobe infiltration, bilateral pedal edema and gynaecomastia are associated with advanced lepromatous leprosy which is rare in children in the 0 – 14 years age group indicating clearly the spectrum is incomplete [22,43]

Indeterminate leprosy [22,44,45,46]

The classic early skin lesion is that of indeterminate leprosy which is most commonly found in the face, extensor surface of limbs, buttocks or trunk. Scalp, axillae, groin and lumbar skin tend to be spared.

Indeterminate lesions consist of one or more slightly hypo pigmented or erythematous macules one or few centimeters in diameter with poorly defined margins. There maybe mild dryness and slight anesthesia maybe demonstrated but usually absent. Sometimes a thickened nerve is palpable.

Peripheral nerves are usually not thickened. This type of leprosy is rare in the extreme of age groups (below 4 years and above 45 years) and commonest age group is 5 – 9 years. [44]

In most instances spontaneous healing occurs.

CLINICAL FEATURES – RIDLEY JOPLING

S. No	CRITERIA	TT	BT	BB	BL	LL
01	Number of lesions	1-3	Multiple	Multiple	Multiple	Generalized
02	Size	Variable	Variable	Variable	Variable	Small
03	Margins	Well defined	Mostly well defined well ill defined	Well –ill defined	Well – ill defined Mostly ill defined	Ill defined
04	Surface	Very Dry	Dry, rough	Smooth, soft Slightly shiny	Smooth, shiny, soft	Smooth, shiny, soft
05	Central healing	Anesthesia in the central healing area	+/-	+/-	+/-	-
06	Satellite	None	+/-	+/-	+/-	-
07	Sensation in lesions (not face)	Absent	Moderately – markedly diminished	Slightly – moderately diminished	Slightly diminished	Not affected
08	Loss of hair over the lesion	Absent	Markedly diminished	Moderately – diminished	Slightly diminished	Not affected
09	Loss of sweat	+	+/-	+/-	+/-	+/-
10	Local cutaneous nerve	+	+/-	-	-	-
11	Symmetry	-	-	-	-	+
12	Nerve involvement	Nerves close to the skin lesion may be affected	Multiple nerves affected	Multiple nerves affected	Multiple nerves affected	Multiple nerves affected
13	Nerve Damage	Nil	Common	Common	Common	Common in advanced stages
14	Other Systems Nose Larynx Eyes Testis Gynaecomastia	 - - - - -	 - - - - -	 - - - - -	 Mild +/- +/- +/- -	 Severe + + + +
15	Bacteriological	-	+/- + from lesion	+	++	+++
16	Immunological - lepromin test	+++	+	-	-	-

Tuberculoid leprosy(TT)

The condition may be purely neural with pain and swelling of the affected nerve, loss of sensation, tingling, muscle weakness and paralysis. Alternatively skin lesion appears with or without nerve involvement.

The typical lesion is a plaque, less commonly a macule, erythematous or copper colored, hypopigmented in dark skin with raised and well-defined edges and a tendency to central flattening. The surface is dry and hairless, insensitive and sometimes scaly. It is usually single but there may be two or three and size seldom over 10 cms in diameter.

Another characteristic feature is the thickening of cutaneous nerve supplying the area or a thickened nerve may be felt in the vicinity.

The extent of large peripheral nerve involvement and disability due to it is minimal or nil in tuberculoid. Nerve thickening may be smooth or irregular and rarely a cystic swelling – cold abscess of nerve and even more rare is calcification of nerve.

Nodular leprosy in childhood is a benign clinical variant of tuberculoid leprosy that affects the breast-feeding infants and children exposed to a highly infected environment such as those born to lepromatous parents, living in leprosy sanatorium, characterized by indurated papulo nodules, nodules, weal like lesions, raised macules, solitary infiltrations and lichenoid lesions usually on the cheeks, arms, limbs, buttocks which resolves spontaneously without any peripheral nerve involvement or deformity considered to be a manifestation of allergy and congenital immunity to Mycobaterium leprae. [50]

Minor tuberculoid: The lesion are small, only moderately elevated often at the margins showing the characteristic papules at the periphery (pebbling) which proceed to rapid central clearing and not associated with severe nerve involvement.

Major tuberculoid: Large lesions, which tend to be more numerous and more markedly and more uniformly raised and are often associated with severe nerve involvement. These lesions may also involve the spared areas especially the scalp, axillae, groin, palms and soles [49]

The clinical descriptive terms major and minor tuberculoid refer to lesions in borderline tuberculoid patients.

Borderline tuberculoid (BT)

Borderline tuberculoid is the most common type seen in children. [6,15,26,42,51,52] The skin lesions of borderlne tuberculoid retain more characters of tuberculoid leprosy. It may be present as macules or plaques or both. The number of lesions is greater than TT, up to 10 or 20 or more and it is asymmetrical. They vary in the size and may be large enough to embrace the whole limb.

Satellite lesions lie near the edges of larger lesions. Margins may be raised and well defined in part of a lesion, flat and vague in another. Hypopigmentation, dryness pebbling and scaling are less pronounced than in true TT, and there is less anesthesia in the lesions.

Peripheral nerve damage is widespread and severe. Peripheral nerve involvement is asymmetrical may be irregularly enlarged, tender nodular thickening and frank abscess formation may occur.

The striking feature of BT leprosy is the occurrence of high frequency of type 1 reaction. Nerve function may deteriorate rapidly and irreversibly leading to various deformities

BT leprosy may present with large pale macules and multiple nerve involvement, it is sometimes called maculoanesthetic or low resistant tuberculoid leprosy associated with liability to severe reaction.

Borderline Borderline (BB)

Most unstable part of the spectrum is occasionally seen in children.

The skin lesions are more in number but not as many as in lepromatous leprosy. There is often a tendency to symmetry. The skin lesions may be macules, papules or plaques or a combination of all. They vary greatly in size, shape and distribution and edges may be well defined in one area and vague in another area. Satellite lesions are common. Geographic appearance of lesions and lesions with ill-defined sloping outer margin and a punched out center with a very well demarcated edge may characterize this spectrum.

Nerve damage is variable. It may be asymmetrical multiple mono-neuropathy if the patient is downgrading from BT to BB or may be symmetrical polyneuritis if the patient is upgrading from BL to BB.

Glove and Stocking sensory loss do not occur.

Borderline lepromatous(BL)

This group shows more of lepromatous characters but still shows few tuberculoid features, occasionally seen in children. This starts with vague macules, initially may be a small group but soon become widespread symmetrically over the trunk. They may be hypopigmented, erythematous or shiny. The macules of BL are more distinct, smaller more variable in shape and also perfectly symmetrical. Loss of sensation, decreased sweating and hair growth start sooner in BL than LL. As the disease progresses some of the macules becomes infiltrated usually centrally. Papules and nodules may develop which are more defined and less symmetrical than those of LL.

Peripheral nerve involvement is widespread occur sooner than in LL though not so symmetrical and signs of damage occur soon. Nerves are less commonly tender than in BT because spontaneous reactions are less common.

Many patients reach BL having down graded from BT. These patients have many features of BT like large macule, circinate lesion with some central healing, but the spectrum may be suspected for the extent, number and small size of majority of skin lesions and the fact that infiltration is central rather than peripheral especially in newer lesions. Nerves may be grossly enlarged and irregular and there may be extensive paralysis and anesthesia from previous type I reaction.

Patients with BL do not suffer from the extensive consequences of bacillary multiplication that are seen in LL. Glove and stocking anesthesia, corneal anesthesia and madarosis are less marked. Type II reactions occur in 25% of the patients. Reversal reactions may also occur.

Lepromatous leprosy(LL)

This entity is sparingly seen in children. An occasional case of indeterminate leprosy may directly land into lepromatous leprosy in children. Clinical features largely correspond to those seen in adults. Glove and stocking anesthesia, corneal anesthesia, madarosis, leonine facies and various systemic involvement are characteristic.

The skin lesions may be macular, papular, infiltrated and nodular. Rarely ulcerative lesions may also occur. The early lesions of lepromatous leprosy are usually macules that are widely and symmetrically distributed. They are usually ill defined, slightly hypopigmented and slightly erythematous. The surface may be shiny and moist. As they progress the entire body surface tends to be involved, but on careful examination less change will be found in the skin in warmer areas such as the axillae, the mid line of the back, the perineum, groin and scalp. Sensation to touch and pin prick is usually unimpaired in early lepromatous macules, but sweating may be diminished.

If the patient is not treated at this stage the skin becomes more and more infiltrated, and takes on a waxy appearance. Skin creases are lost and erythema increases. The distribution is characteristic: on the face over the forehead, zygoma, the chin and the ear lobes, and on the limbs over the cooler dorsal

areas, the fore arms, back of the hands and the extensor surfaces of the lower legs. By this stage there is clinical evidence of nerve damage. First there is loss of sensation over the dorsum of the hands, forearms and lower legs. The area of sensory loss spreads slowly until all the skin is anesthetic except the axillae, groin and scalp. Only from these warm areas the patient can sweat, and on hot days he may be distressed by the profuse sweat that occurs in them and may lead to fatal hyperpyrexia.

Hair is lost in all skin lesions, especially on the face. Loss of eyelashes and eyebrows – madarosis, is characteristic. The scalp hair is usually spared. Very rarely, in advanced disease, there may be residual hair growing only in bands over the course of the arterial supply to the scalp called leprous alopecia. Nail growth may be disturbed late in the course of the disease and nails may lose their luster and become thin, ridged and curved. Infiltration heaps up the skin of the face into great folds, producing the leonine facies.

There is gradual appearance of sensory and automatic nerve damage in the cooler parts of the skin, but it may be difficult or impossible to find clinical signs of damage to the large peripheral nerves until the disease is well advanced. As the disease progresses the peripheral nerves first become firm, then enlarged, then hard, at sites of predilection, symmetrical. Muscular weakness sometimes appear a little sooner, possibly because the muscles of the hand the feet are affected directly as well as through the peripheral nerves. Glove and stocking anesthesia is a characteristic

Involvement of nasal mucosa is commonly seen in 80% and carries a high risk of infectivity. The patient may develop stuffy or blocked nose,

mucopurulent discharge and epistaxis. The mucosa of nasal septum and inferior turbinates looks yellow and swollen and covered with crusts.

Involvement of the larynx is a late manifestation and it may be a fibrotic or an ulcerative form.

Involvement of eyes may be due to, abnormal exposure of the eyes secondary to involvement of the fifth and seventh cranial nerves, infiltration of the eyes and the surrounding tissues by the leprosy bacilli, inflammation of the eyes secondary to the infiltration by the leprosy bacilli and complication of the eyes secondary to involvement of surrounding tissues: eyelids, lacrimal glands and the nasolacrimal drainage system.

Involvement of the internal organs, liver, spleen, kidney and adrenals may occur but testicular involvement is common. Gynecomastia may follow testicular atrophy. Muscles and lymph nodes are also involved.

Bone and joint involvement ranges from mild tenosynovitis to leprous osteomyelitis.

Pure neuritic leprosy

Primary neuritic leprosy occurs in children though less commonly than in adults. [(53)] Primary neuritic leprosy usually present with signs and symptoms of nerve deficit. This may be a gradual weakness in a hand or a sudden foot drop or it may present as anesthesia in an extremity or extremities. On examination the relevant peripheral nerves and sometimes others are enlarged. If they are in reactional state, the nerves will be tender to palpation or

spontaneously painful. The diagnosis is usually made by the presence of definite nerve enlargement. The symptoms caused by the affected nerves include sensory, motor and trophic changes in the area supplied by the nerves. These may result in deformities neuropathic ulcers and lagophthalmos which may result in severe eye complications.

A spectrum of TT to BL has been observed in histopathology of pure neuriitic leprosy. The ulnar, median, common peroneal, the posterior tibial, the greater auricular and rarely radial nerves are involved in the order of frequency. The cranial nerves involved are 5^{th} and 7^{th}. Abscess formation when observed suggests a tuberculoid histology.

Above 15 – 35 % of pure neuritic leprosy patients develop skin lesions during follow up. (54)

Histoid leprosy

Unusual variants like histoid leprosy are rare in children. Histoid leprosy most commonly occurs in 20 –40 years age group but it has been reported in children as early as 9 years, 10 years and in an 8 year old child whose mother is a case of lepromatous leprosy. (55)

Histoid leprosy appear as cutaneous / sub-cutaneous, firm, translucent, erythematous / coppery, shiny papules, nodules or plaques emanating from an apparently normal skin. The lesions are usually located on the back, buttocks, face and extremities and over bony prominences especially around the elbows and knees. It usually follows dapsone monotherapy. (56)

Ocular leprosy [57]

Ocular leprosy among children is rare and non-blinding. Lagopthalmous, patch around the eye. Nodules and anesthetic cornea are common presenting symptoms in children.

Genital involvement

Occurrence of lesion over genitalia both in tuberculoid and lepromatous pole has been reported in children. The youngest being a 4 year old in a case of borderline lepromatous [16] and penile tuberculoid leprosy in a 5 year old boy whose father is a case of lepromatous leprosy. [58]

Reactions

Reactions were not uncommon in children. [6,15,42] Predominantly type I reaction is seen than type II reaction. [6,15,42] Both type I and type II reaction has also been encountered. [42] Reaction may be a presenting feature in children. [42] There are no reports of reaction (both type I or type II) in cases below 5 years age. [16] The criteria for diagnosis of reaction shall be the same for adults.

Type I Reaction

It is a delayed hypersensitive reaction due to rapid change in cell-mediated immunity. It is typically seen in borderline patients because of the immunological instability. It may be upgrading (reversal) or downgrading reaction. Upgrading reaction occurs in patients when the immunological status shifts towards tuberculoid spectrum, usually during the first six months of

treatment. Downgrading reaction occurs when there is deterioration of immune status and the patient shifts towards lepromatous pole.

The most prominent sign is the rapidly developing change in the appearance of some or all the skin lesions; they become erythematous, more prominent, shiny, warm to touch, and resembling erysipelas. The lesions are often tender and painful. Sometimes necrosis supervenes with breakdown and ulceration. Lesions desquamate as they subside. New lesions may appear. Usually the new lesions resemble the pre-existing ones but it may be numerous and small, and in case of downgrading reaction, the new lesions may be more lepromatous in appearance.

Neuritis is the most important part of type I reaction. Neuritis presents as enlargement of one or more nerves with pain and tenderness at the sites of predilection. Anesthesia develops rapidly in the distribution of the affected nerve. More severe motor disturbances like claw hand, foot drop, facial palsy may occur. Rarely nerve abscess may occur. Another associated manifestation is edema of hands, feet, or face; sometimes all three sites are involved, or, rarely, one foot or hand. Constitutional symptoms are rare.

Type II Reaction

It is an immune complex syndrome (type III hypersensitivity reaction). It occurs almost exclusively in lepromatous leprosy, only occasionally appearing in borderline lepromatous leprosy. There is no change in the appearance of the leprosy lesions, but there is occurrence of crops of brightly erythematous nodules, which come and go. Systemic disturbance is usual.

Unlike upgrading reaction, when it occurs in relation to therapy, it is very unusual for it to occur during the first six months of therapy. It tends to occur later during the course of treatment when the skin lesions appear quiescent and all or most of the bacilli in the skin are granular, however patient may be in reaction when first seen.

Erythema nodosum leprosum(ENL) lesions are brightly erythematous, slightly raised nodules or plaques, variable in size but usually small, and if multiple tend to be distributed bilaterally and symmetrically. They are often tender, warmer than the surrounding skin, blanch with light finger pressure and evanescent. They commonly occur on the face, arms and thighs; the flexor aspects of the forearms and the medial aspects of the thighs are favoured, rarely on the palms and soles. They fade leaving a blue stain. When ENL lesions are numerous there is likely to be fever and malaise. The fever being intermittent with its fastigium in the evenings, and it is usual to find fresh crops of ENL lesions appearing between 17.00 and 18.00 hours, a time when endogenous cortisol production is at its lowest. They desquamate as they subside. In severe Type 2 reaction, ENL lesions may become vesicular and bullous.

Type 2 reaction may be associated with nerve pain, periosteal pain, muscle pain, pain and swelling in joints, rhinitis, epistaxis, acute iritis, painful dactylitis, swollen and tender lymph nodes, acute epididymo-orchitis, and proteinuria. The face and hands and feet may become edematous and the spleen may become palpable.

Deformities and disabilities

It is well established that deformities are uncommon in children. The low incidences of deformities in children are due to mild form of disease, shorter duration, and tendency towards self-healing and low incidence of reactions. [59]

Both paresis and established deformities is more common in BT leprosy and established deformity in lepromatous pole. Left hand deformities are more common in children in contrast to right hand involvement in adults due to labour. [59] Claw hand is the more common deformity followed by trophic ulcer, foot drop and wrist drop. [26]

Relapse

Relapse is defined as a return of active disease in a patient who has apparently completed a prescribed course of treatment and whose treatment was therefore stopped by an authorized member of the health services. Early relapse is within 3 ½ years and is due to insufficient treatment, insufficient drugs, improper classification and insufficient duration. Late relapse is after 3½ years and is due to bacterial persistence and drug resistance.

The features of relapse are extension in area of the existing lesions, thickening, erythema or infiltration of previously subsided lesions, or new lesions; thickening and tenderness of nerves and/or fresh nerve involvement and bacteriological positivity in previously negative sites and/or positivity in fresh lesions.

Relapses were not uncommon in children. It usually occurs after treatment with paucibacillary regimen. [15]

Differences between reversal reaction and relapse

Reversal reaction	Relapse
Seen in BT, BL, LLs types	Relapse may be seen in all types.
Onset is sudden, within 6 months of termination of treatment.	Onset insidious, often after 6 months following termination of treatment
Swelling, erythema, and scaling of lesions. Rarely new lesions of same morphology. Tenderness of lesion on tapping or on pressure between two fingers. Edema of feet and hands. Occasionally ulceration of lesions	Old lesions show extension in area with increased signs of activity, new lesions appear, edema is not a prominent feature, change in type may be seen.
Previously involved nerves may be tender, with sensory and/or motor deficit deterioration. Nerve abscess may form.	Fresh nerves involved. Nerves show thickening and tenderness on deep pressure. Sudden onset of paralysis is not seen.
Skin smears usually negative, Bacteriological index is lowered in BL	Skin smear may be positive, bacteremia may be seen.
Response to steroids good	Steriod therapy not very effective.
Subsidence within 2 months	Progressive under steroid.

Natural evolution of disease in children [9,22]

The course of untreated leprosy in children is variable and to a great extent unpredictable. Progression and regression of lesions is common and new lesions can appear and old once disappear in a period of months or a few years.

Transformation of one type of lesion into another is also frequent. Spontaneous regression of lesions occurs in about 75 % of cases and only about 6 % of children had active leprosy into adulthood. [22]

The prognosis of maculoanesthetic type is good and much better for indeterminate lesions. The lesions may heal or pass onto tuberculoid type and only rarely pass onto lepromatous pole. [9]

Prognosis [9,22]

Spontaneous regression occurs in about 75% of cases. In children regularly treated the prognosis of tuberculoid and indeterminate cases is excellent. The progression of clinical signs is almost irreversibly halted and lesions subside leaving few sequelae such as atrophy, scar or a hypopigmented area with some loss of sensation.

The distressing sequelae due to neural involvement and reactional episodes are mostly due to delayed and injudicious treatment. As lepromatous leprosy and borderline lepromatous leprosy cases are comparatively rare among children, the overall prognosis in childhood leprosy is very good.

HISTOPATHOLOGY OF SKIN [60,61,62,63]

Indeterminate leprosy

The epidermis may show areas of atrophy but usually normal. The inflammatory infiltrate consists of histiocytes and mainly lymphocytes usually involve only 10 % of dermis or less. The inflammatory cells present both in superficial and deep dermis surround the neurovascular bundles, superficial

deep dermal vessels, sweat glands, arrector pili muscle and other skin adnexa. Nerve bundles are selectively involved and lymphocytes and histiocytes may infiltrate the perineurium and endoneurium. Schwann cell hyperplasia is a feature. Granuloma formation is never seen. Although the presence of selective preineurial and endoneurial inflammation is highly suggestive of leprosy a definitive diagnosis of leprosy can be made only on finding acid - fast bacilli (AFB) in any one of the following locations, immediately underneath the epidermis, within nerve bundles, arrector pili muscle or in a macrophage around a vessel demonstrated by Fite Faracco stain.

Tuberculoid leprosy

Atrophy of epidermis and some areas of minimal hyperplasia. Discrete and confluent granuloma in dermis sometimes occupying about entire dermis and hugs the epidermis and erosion of segment of epidermis may be seen. Granulomas extend along neurovascular bundles and are composed of well-formed tubercles with central collection of epithelioid cells and few Langhan's giant cells surrounded by dense infiltrate of lymphocytes always involving dermal nerves. The adnexa and arrector pili are infiltrated and destroyed. Sub-epidermal zone, fibrinoid and caseous necrosis and acid - fast bacilli are absent.

Borderline tuberculoid

Epidermal atrophy over areas where the dermal granuloma occupies a major portion of the dermis. There is a clear sub epidermal zone. The granulomas are composed of poorly formed tubercles with epithelioid cell collection and occasional Langhans' giant cells and mixed with lymphocytes.

The granuloma may extend towards the epidermis and occupy the sub epidermal zone in focal area, rarely. The nerves show intra and perineurial granuloma. Nerve destruction, leaving behind fragments of nerves is also seen. Widespread damage to adnexal structures is present. There is no caseous or fibrinoid necrosis. Few AFB is seen inside nerve bundles, arrector pili muscles or in a macrophage in the granuloma.

Borderline borderline leprosy

The epidermis is atrophic and is separated from the granuloma by a clear grenz zone. The granuloma is composed of large collections of plump epithelioid cells, some macrophages, scanty lymphocytes and no Langhans' giant cells. Nerve bundles are surrounded and partly destroyed by granuloma. There is reactive proliferation of perineurial cells and edematous thickening of the perineurium. A few lymphocytes are seen inside the nerves. Small numbers of AFB are present within nerve bundles and in the macrophages scattered among the epithelioid cells.

Borderline lepromatous leprosy

The epidermis is atrophic and clear zone separates the epidermis from the granuloma. The poorly formed granuloma consists of macrophages and numerous lymphocytes and plasma cells.

The macrophages may have a foamy cytoplasm but not pronounced. Scattered small collections of epithelioid cells are present. The granuloma surround adnexal structures and nerve bundles. The perineurium develops

marked laminations giving rise to a typical onion peel appearance and is also infiltrated by many macrophages and lymphocytes.

Clumps of AFB are identifiable within macrophages, perineurial cells, endothelial cells, Schwann cells and arrector pili muscles.

Lepromatous leprosy

The epidermis is atrophied and thin. The rete ridges are completely flattened. There is a clear grenz zone. Most of the macrophages initially have a pink and granular cytoplasm as the lesions grow older the cytoplasm becomes foamy and vacuolated – the so-called Virchow cells and lepra cells. There are only few lymphocytes scattered among the macrophages. Focal collections of plasma cells are distributed throughout the inflammatory lesion. The cellular infiltrate may be seen as a small focal clusters in the dermis in early lesions, and as the disease progress these focal infiltrates merge together to form a band of macrophages infiltrating the dermis and extending to the subcutaneous fat. The skin adnexa are surrounded by macrophage and show signs of atrophy. Nerves also show perineurial collections of macrophages. There is only minimal reactive proliferation of the perineurium and the amount of intraneural infiltration by inflammatory cells is insignificant.

Acid-fast stain shows clumps of bacilli in Schwann cells, perineurial cells, macrophages endothelial cells, arrector pili muscles, sweat and sebaceous glands and hair follicles.

TYPE I REACTION

Upgrading reaction

The most important characteristics feature is edema, which is intense during the acute phase. The inflammatory cells in the lesion are spread out and there is disorganization of granuloma. Some nerves may undergo caseous necrosis. Rarely a few scattered neutrophils are seen. There is a well marked increasing lymphocytes. As the reaction subsides there is reduction in the edema and there is formation of well-organized tubercles. Many Langhans' giant cells may also be seen. Acid-fast bacilli in the lesion are considerably reduced or completely disappeared.

Downgrading reaction

There is edema, reduction in lymphocytes and there is appearance of increasing number of macrophages with AFB. Sometimes Langhans' giant cells may persist in these lesions.

Type II reaction. (Erythema nodosum leprosum)

There is dense infiltration of superficial and / or deep dermis and / or subcutaneous tissue by neutrophils. They are superimposed on an already existing lepromatous granuloma. Often the influx of neutrophils is so intense to form microabscesses. Vasculitis is a predominant feature in some cases. Damage to collagen and elastic fibres is common. In necrotising ENL there is also necrosis and ulceration of skin. There is a local reduction in the bacterial

load and most of the organisms present are broken and granular. During the healing phase the neutrophils are gradually replaced by lymphocytes.

Leprous neuritis [64,65]

Tuberculoid neuritis

There is increased vascularity of epineurial connective tissue, which may contain occasional perivascular tuberculoid granuloma. The perineurium shows thickening and lamination due to proliferation of perineurial cells and fibrous tissue and is also often infiltrated by a granuloma. All nerve fascicles of a nerve may be infected with every fascicle showing perineurial thickening, endoneurial infiltration and replacement of the nerve parenchyma by granulomas, or, there may be selective involvement of fascicles with one or a few fascicles or only a small part of fascicles being involved. A fairly normal looking fascicle may be seen alongside one infiltrated by a granuloma and replaced by fibrous tissue. The granuloma consists of epithelioid cells, lymphocytes and occasional Langhan's giant cells. There may be small focal areas of caseous necrosis. The affected nerve parenchyma is usually irreversibly destroyed. In focal areas the granuloma sometimes invades the surrounding tissues penetrating the perineurium. A few acid-fast bacilli (AFB) are found in caseous material and in Schwann cells. During healing, the markedly thickened and lamellated perineurium and destroyed endoneurium are replaced by hyalinized fibrous tissue.

Nerve abscess in tuberculoid is a well-known feature. It consists mostly of caseous necrotic material surrounded by tuberculoid granuloma and fibrous

tissue. Remnants of nerve may be detected at the periphery of the granuloma. AFB are often seen in the necrotic material in a majority of patients.

Borderline neuritis

In BT leprosy histologically the appearance is somewhat similar to tuberculoid leprosy. There is granulomatous destruction of the nerve that is irreversible.

In BL leprosy there is marked infiltration of the perineurium and endoneurium of the nerve by lymphocytes and foamy macrophages.

Lepramatous neuritis

All the fascicles of the nerve is involved with fibrous thickening of the epineurium and perineurium. The endoneurium looks almost normal except for edema and infiltration by scattered lymphocytes. Acid-fast stain shows clumps of bacilli inside Schwann cells of an almost normal looking nerve. In patients receiving antileprosy therapy the disease regresses and Schwann cells and macrophages undergo foamy change and the partly destroyed nerve is replaced by fibrous tissue. Acid-fast stain shows the foamy macrophages and Schwann cells to contain numerous fragmented and granular AFB some of which are in clumps. In untreated patients the disease progresses and the nerve parenchyma may be extensively replaced by granulomas composed of macrophages , lymphocytes and fibrous tissue. Acid-fast stain shows large clumps of AFB many of which are solid staining inside macrophages and Schwann cells. With time fibrous tissue gradually replaces the granulomas and nerve tissue. Focal intraneural collection of foamy macrophages may persist long after the skin

smears have become negative. Rarely a few a granular AFB may be demonstrated in such nerves many years after the patient declared cured.

Neuritis in pure neural leprosy

The histological changes found in pure neural leprosy is often that of BT or BL leprosy and only occasionally TT or LL types are seen. Pure neural leprosy lesions are easily mistaken for neuropathies other than leprosy, therefore a suitable nerve biopsy is mandatory for diagnosis.

Neuritis in reaction

Reversal reaction (Type I) shows active destruction of nerves by increasing numbers of epithelioid cells and lymphocytes and even caseous necrosis. There is edema followed by increase in intraneural pressure and ischaemic changes.

ENL (type II reaction)

There is edema and infiltration with large numbers of polymorphs. The nerve parenchyma is destroyed by enzymes released from neutrophils and by ischaemic changes caused by an increase in intraneural pressure.

End stage neuritis

In all type of leprous neuritis as the lesion resolves, the perineurium and endoneurium are replaced by hyalinized fibrous tissue and it is often difficult to differentiate it from healed nerve lesions from other causes. The features found in a fascicle which may be helpful in identifying healed leprous neuritis or a

few AFB persisting inside dense fibrous tissue, small collections of foam cells persisting in a fibrosed hyalinized nerve, caseous necrosis surrounded by dense fibrous tissue and severe lamination of the perineurium with many layers of hyalinized fibrous tissue.

AIM OF THE STUDY

This study was done to find out the following in childhood leprosy 0 to 14 years age group.

1. Common age group of leprosy in children.
2. Sex incidence.
3. Duration of the disease.
4. History of household contact.
5. Clinical presentation – morphology, color, number size and common site of skin lesion, common peripheral truncal nerve and cutaneous nerve involvement.
6. Common spectrum of the disease.
7. Reactions.
8. Deformity.
9. Slit skin smear.
10. Histopathological examination of skin and nerve biopsy.

MATERIALS AND METHODS

Childhood leprosy cases from 0 to 14 years age attending the Department of Dermatology and Leprosy of Government General Hospital, Chennai were collected from August 2004 to August 2006.

A complete history of presenting complaints, duration of illness, history of contact, treatment history, BCG vaccination was taken from the informant, usually the parents. A complete general examination and dermatological examination regarding the morphology, number, size, site, color, anesthesia, margins, surface, satellite lesion and central clearing of skin lesions and involvement of peripheral truncal nerve and cutaneous nerve was done.

Reactions and deformities were also noted.

Slit skin smear examination was done from a minimum of 3 sites if there was a single lesion, i.e. from the lesion and both ear lobes and from a minimum of 4 sites if there were more than one lesion i.e. from both ear lobes and 2 skin lesions.

Skin biopsy was done from the lesion and stained with haematoxylin and eosin and Fite Farraco staining for histopathological examination.

Nerve biopsy was done and stained with haematoxylin and eosin in cases of pure neuritic leprosy.

OBSERVATION

During the study period of 2 years, total number of childhood leprosy cases 0 –1 4 years age group seen were 46 cases.

AGE AND SEX

Sex	Age group			Total
	0 –4 Years	**5 – 9 Years**	**10 -14 Years**	
Male	2	3	26	31
Female	1	4	10	15
Total	**3**	**7**	**36**	**46**

Most number of cases were seen in 10 – 14 years age group **36** cases (78.3%)

Number of male cases **31**

Number of female cases **15**

Male female ratio **2.07 : 1**

AGE AND CLASSIFICATION

Classification	Age in years			Total	Percentage
	0 –4 Years	5 – 9 Years	10 -14 Years		
Indeterminate	0	1	2	3	6.5 %
Tuberculoid	1	2	4	7	15.2 %
Borderline Tuberculoid	2	3	22	27	58.7 %
Borderline Borderline	0	0	0	0	0
Borderline Lepromatous	0	0	4	4	8.7 %
Lepromatous	0	0	0	0	0
Pure neuritic	0	1	4	5	10.9 %
Total	**3**	**7**	**36**	**46**	**100%**

The most commonly seen spectrum was Borderline tuberculoid leprosy 27 cases (58.7 %).

The youngest case in the study was a case of Borderline tuberculoid leprosy seen in a 4 year old female child with age of onset at 3 years.

Two more children aged 4 years, one a case of Indeterminate Leprosy with age of onset at 3½ years another one, a case of Tuberculoid Leprosy with age at onset of 3 years and 9 months were also seen in the study.

SEX AND CLASSIFICATION

Classification	IND	TT	BT	BB	BL	LL	PNL	Total	Percentage
Male	2	2	22	0	3	0	2	31	67.4 %
Female	1	5	5	0	1	0	3	15	32.6 %
Total	3	7	27	0	4	0	5	46	100 %

Leprosy towards lepromatous pole was found to be uncommon before puberty in both sexes and great majority of cases are towards tuberculoid pole.

AGE AND DURATION OF THE DISEASE

Duration of disease in years	Age in Years			Total
	0-4 Years	5-9 Years	10-14 Years	
≤ 1	3	5	27	35
2 – 3	0	2	7	9
≥ 4	0	0	2	2
Total	**3**	**7**	**36**	**46**

The duration of disease in most of the cases ≤ is 1 year (35 cases).

AGE AT ONSET OF LEPROSY

Classification	Age in years			Total
	0 –4 Years	5 – 9 Years	10 -14 Years	
Indeterminate	0	1	2	**3**
Tuberculoid	1	2	4	**7**
Borderline Tuberculoid	3	6	18	**27**
Borderline Borderline	0	0	0	**0**
Borderline Lepromatous	0	3	1	**4**
Lepromatous	0	0	0	**0**
Pure neuritic	0	1	4	**5**
Total	**4**	**13**	**29**	**46**

The age at onset was calculated by subtracting the duration of disease from the age of the patient. The age at onset for most of the cases is 10 - 14 years. (29 cases).

CONTACT WITH LEPROSY IN THE FAMILY

Household Contact with Leprosy	Number of children
Father	4
Mother	2
Sibling	1
Grand mother	1
Others	1
Total	**9**

Percentage of cases with household contact was found to be 19.6%. Father found to be the index case in most cases.

AGE AND PATCHES

Number of Patches	Age in Years			Total	Percentage
	0-4 Years	5-9 Years	10-14 Years		
0	0	1	4	5	10.9 %
1	1	3	18	22	47.8 %
2	0	0	4	4	8.7 %
3	2	1	1	4	8.7 %
4 – 5	0	0	2	2	4.3 %
> 5	0	2	7	9	19.6 %
Total	**3**	**7**	**36**	**46**	**100 %**

Single skin lesion is the most common presentation in 22 cases (47.8%).

DISTRIBUTION OF SINGLE SKIN LESION

Site of the lesion	**Number**
Face and Neck	
Cheek	5
Chin	1
Total	6
Trunk	
Upper back	3
Total	3
Lower Limbs	
Buttocks	1
Thighs	1
Knees	2
Legs	1
Total	5
Upper Limbs	
Arms	2
Fore Arms	6
Total	8
Total of All	22

The distribution of single skin lesion was more over the exposed parts like fore arms, face, knees and legs - 15 cases (68 %), than over the covered parts like back, arms, buttocks and thighs - 7 cases (32 %) out of the 22 patients presented with single skin lesion.

The common morphology seen were macules, patches and plaques.

Most of the lesions were hypopigmented, followed by erythematous and copper colored lesions.

The size of the lesions varied from less than 1cm to 45 X 15 cms size which was the largest skin lesion seen in the study occupying most part of the lower limb.

The clinical presentation in most of the cases was hypopigmented skin lesions with impairment of sensation. The other mode of presentation was numbness of the hands and feet, foot drop, clawing of the hand and swelling of the nerve (ulnar nerve abscess) in cases of pure neuritic leprosy.

Pattern of nerve involvement

I. **Cutaneous nerve involvement**

Cutaneous Nerve	Unilateral	Bilateral	Total
Greater auricular	3	1	4
Radial Cutaneous	7	6	13
Sural	4	0	4
Supra Orbital	3	0	3
Supra Clavicular	2	0	2
Medial Cutaneous nerve of thigh	1	0	1
Intermediate Cutaneous nerve of thigh	1	0	1
Lateral cutaneous nerve of thigh	1	0	1

Commonest cutaneous nerve involvement was radial cutaneous, followed by greater auricular, sural, supra orbital and supraclavicular. Involvement of medial, intermediate and lateral cutaneous nerves was seen in one patient.

II. Truncal Nerve involvement

Truncal Nerves	Unilateral	Bilateral	Total
Ulnar	22	5	27
Median	5	1	6
Lateral popliteal	17	3	20
Posterior tibial	7	7	14

Commonest truncal nerve involvement was ulnar nerve, followed by lateral popliteal, posterior tibial and median nerve.

Reactions

Type I Reaction was seen in 3 children, of whom 2 had Borderline lepromatous leprosy and 1 had Borderline tuberculoid leprosy.

Type II Reactions was not seen in this study.

Relapse

Relpase was seen in a 10 year old male child, who had presented with an increase in the size of his skin lesion, 2 years after completion of paucibacillary treatment for tuberculoid leprosy. The clinical features and histopathology of the patient was found to be consistent with the borderline tuberculoid.

Deformities

Deformities were seen in 5 children.

1. 14 year old male child, a case of pure neuritic leprosy, had claw hand both sides and trophic ulcer right foot.

2. 14 year old male child, a case of Borderline Tuberculoid Leprosy had left side claw hand and left foot drop.

3. 11 year old male child, a case of pure neuritic leprosy, had clawing right little finger.

4. 12 year old female child, a case of pure neuritic leprosy, had left foot drop.

5. A 6 year old female child, a case of pure neuritic leprosy, had left foot drop.

Special variants like histoid leprosy was not seen. Ocular involvement was not seen in any case. The frequently encountered differential diagnosis were polymorphic light eruption, pityriasis alba, tinea versicolar, early vitiligo, resolving morphea and post inflammatory hypopigmentation. In cases of poly neuritic leprosy, the differential diagnosis encountered were trophic ulcer following trauma to the knee and subsequent neurological deficit and type I hereditary sensory autonomic neuropathy. A case of ganglion, mimicking nerve swelling, along the course of larteral popliteal nerve, was also seen.

Slit skin smear was positive (Bacteriological Index - 4+) in all the 4 borderline lepromatous cases and negative in the rest of the cases.

Skin biopsy was done in all the cases and stained with haematoxylin and eosin and the histopathological features were consistent with the clinical type.

Fite Farraco staining was done in all the cases and found to be positive for acid-fast bacilli in Borderline Lepromatous cases and negative in rest of the cases.

Nerve biopsy in cases of pure neuritic leprosy showed tuberculoid granuloma.

DISCUSSION

The total number of childhood leprosy cases (0 to 14 years age group) seen was forty six during the study period of two years.

Age Distribution

Of the forty six patients, 36 (78.3%) children were in the 10-14 years age group, followed by 7 (15.2%) children were in the 5-9 year age group and 3 (6.5%) children in the 0 - 4 years age group. Age distribution reflected a clear preponderance of older children, 10 - 14 years age group, as seen in previous similar studies. This could be because of a relatively long incubation period for this slow infection.

The youngest patient seen in the study was a 4 year old girl who had Borderline tuberculoid leprosy with the age at onset of 3 years of age. Two more children of 4 years old, one with indeterminate leprosy and another with tuberculoid leprosy, with age at onset of 3 years 6 months and 3 years 9 months respectively, was also seen in the study.

The age at onset of leprosy in most of the cases , 29 cases (63%) was 10 - 14 years among, the children in the study group (0-14 years).

Sex Distribution

There was a preponderance of boys 31 cases, over girls 15 cases, with a male : female ratio of 2.07 : 1. The preponderance of male children in the study is similar to the observation is most of the earlier studies. This could be

because of a number of environmental and socio-cultural factors such as a greater exposure in boys.

Duration of disease

The duration of disease at the time of detection (as stated by parents) in most of the cases, 35 cases (76.1%) was found to be ≤ 1 year.

House hold contact

Over all, nine children (19.6%) gave a history of contact with a leprosy patient within the household, the father being the index case in 4 cases followed by the mother in 2 cases.

The duration of illness and the incidence among household contact in childhood leprosy patients is comparable with earlier studies.

Spectrum of the disease

The commonest spectrum seen is the borderline tuberculoid, 27 cases (58.7%). It was well established that borderline tuberculoid is the commonest spectrum among children in almost all the previously conducted studies.

Only four cases of borderline lepromatous leprosy were seen and no lepromatous leprosy cases were seen indicating clearly the spectrum is incomplete. It is a paradox that children, who have poor cell-mediated immunity, rarely present with multibacillary disease.

Pure neurtic leprosy was seen in five children, of these two children presented with foot drop, one child with numbness below the right knee, one

child with claw hand and trophic ulcer and one child with ulnar nerve abscess and clawing of the right little finger.

Clinical presentation

The clinical presentation in most of the cases was hypopigmented skin lesions with or without impairment of sensation.

Single skin lesion was the commonest presentation seen in 22 (47.8%) cases.

The site of the single skin lesion in most of the cases was the exposed parts like fore arm, face, knees and legs, rather than the covered parts like back, arms, gluteal region and thighs. Some of the earlier studies observed an increased incidence of single skin lesion over the gluteal region. In this study, among the 22 patients presented with single skin lesion, 6 children had lesions over the fore arm, the commonest site, followed by, the cheeks in 5 cases.

The common morphology of the lesions seen were macules, patches and plaques. The lesions were usually hypopigmented, followed by erythematous and copper coloured lesions. The size of the lesions varied from less than 1 cm to 45 x 15 cm sized lesion involving most part of the lower limb, the largest lesion seen in the study. Icthyosis and traumatic fissures were seen in few cases.

Of the peripheral truncal nerve, ulnar nerve is the commonest nerve involved followed by lateral popliteal, posterior tibial and median nerve. The radial cutaneous nerve is the commonest cutaneous nerve involved followed by

greater auricular, sural, supraorbital and supraclavicular nerves. Involvement of the lateral, intermediate and medial cutaneous nerves of thigh was seen in one case.

Reaction, Relapse and Deformity

Type I reaction was seen in three cases and Type II reaction was not seen.

Relapse was seen in a case of a 10 year old boy, who presented with increase in size of his skin lesion, 2 years after completion of paucibacillary treatment.

Deformities were seen in 5 children. Claw hand, foot drop, and trophic ulcer were the deformities encountered.

Ocular involvement was not seen in any of the cases. Special variants like histoid leprosy was also not seen.

Slit skin smear and histopathology

Slit skin smear was found to be positive only in four cases, all of them were borderline lepromatous cases, and negative in the rest of the cases.

Histopathological examination of the skin and nerve biopsy was very useful in establishing the diagnosis.

CONCLUSION

Leprosy in children

- The commonest age group of occurrence is 10 to 14 years.
- Incidence is more among male children than female children, because of increased exposure of boys, due to cultural and socio-economic factors.
- Duration of disease is usually $\leq$ 1 year.
- Father is the most common index case among household contacts.
- Single skin lesion, which occurs predominantly over the exposed parts, is the most common presenting feature.
- Ulnar nerve is the most commonly involved truncal nerve and radial cutaneous nerve is the most commonly involved cutaneous nerve.
- Borderline tuberculoid is the commonest spectrum.
- Leprosy towards lepromatous pole is rare among children, indicating clearly the spectrum is incomplete.
- Pure neuritic leprosy occurs in children in a sizable proportion.
- Reactions, predominantly Type I reactions, relapse and deformities do occur in children, even though rare.

- It is very difficult to demonstrate the acid-fast bacilli by slit skin smear in tuberculoid spectrum, hence histopathological examination of skin and nerve biopsy is ideal in establishing the diagnosis.

- Leprosy still occurs in children in a sizable and constant proportion, even though the prevalence rate has reduced below 1 per 10000 and we are in the run for total eradication of leprosy.

REFERENCES

1. Jopling WH, McDougall. Definition, Epidemiology and World Distribution Hand book of Leprosy, Fifth edition. CBS Publishers and Distributors 1996: 1.

2. Lowe J. Comments on the history of leprosy. Leprosy Review 1947; 18: 54 - 63

3. Jopling WH, McDougall Definition, Epidemiology and World Distribution Hand book of Leprosy, Fifth edition. CBS Publishers and Distributors 1996: 6 – 7.

4. Stephen R Ell, Leprosy in history. In Hastings RC,ed. Leprosy, second edition, Edinburgh: Churchil livingstone, 1994: 3 – 4.

5. John R. Trautman, The history of leprosy. In Hastings RC,ed. Leprosy, second edition, Edinburgh: Churchil livingstone, 1994: 20 – 21.

6. Chander Grover, Soni Nanda, Vijay Kumar Grag, Reddy BSN. An epidemiological study of childhood leprosy from Delhi. Pediatric Dermatology 2005; 22 (5): 489 – 490.

7. Agarwal SP. Final push for elimination of Leprosy in India. Indian Journal of Leprosy; 77(3) 2005: 213 – 215.

8. Rao CK. Leprosy elimination in India – so near. Indian journal of leprosy; 77(3) 2005: 207 – 211.

9. Dharmendra, Ganapati R. Leprosy in children. Studies on leprosy by Bombay leprosy project; 1976 – 1986: 10- 31.

10. Trautman JR Epidemiologic aspects of Hansen's disease. Bull New York Acad Med 1984; 60:722.

11. Jesudasan K, Bradley D, Smith PG, Christian M. Incidence rates of leprosy among household contacts of "Primary Cases". Indian journal of leprosy 1984; 56 (3): 601.

12. Michel F Lechat, Etinenne E. Declercq, Control programs in leprosy. In Hastings RC,ed.Leprosy, second edition, Edinburgh: Churchil livingstone, 1994 : 368.

13. Swain JP, Mishra S, Jena S, Prevalence of leprosy among household contacts of leprosy cases in western Orissa. Indian journal of leprosy 2004; 76 (1): 19 –27.

14. Ebenezer L, Arunthathi S, Kurian N. Profile of leprosy in children: past and present. Indian journal of leprosy 1997; 69(3): 255 – 259.

15. Abraham Selvasekar, Joseph Geetha, Kurian Nisha, Manimozhi N, Jesudasan K and Rao PSS. Childhood leprosy in an endemic area. Leprosy review 1999; 70(1): 21 – 27.

16. Ritika Gupta, Archana Singal & Deepika Pandhi. Genital involvement and type I reaction in childhood leprosy. Leprosy review 2005; 76 (3): 253 –257.

17. Girdhar BK. Skin to skin transmission of leprosy. Indian Journal of Dermatology, Venereology and Leprology 2005; 71(4): 223 – 225.

18. Jasmita Satapathy, Bikash Ranjan Kar, Job CK. Presence of Mycobacterium leprae in epidermal cells of lepromatous skin and its significance. Indian Journal of Dermatology, Venereology and Leprology 2005; 711(4): 267 – 269.

19. Noordeen SK. The epidemiology of leprosy. In Hastings RC,ed. Leprosy, second edition, Edinburgh: Churchil livingstone, 1994: 29 – 44.

20. Prasad PVS. Epidemiology. In All about leprosy, First edition, Jaypee Brothers, Medical publishers (P) Ltd, New Delhi 2005: 41 – 43.

21. Sehgal VN, Koranne RV, Sharma AK, Misra S, Jain RK. Age-at-onset of Leprosy. Leprosy in India 1982; 54(2): 332 – 337.

22. Noussitou FM, Sansarricq H, Walter J. Leprosy in children. Geneva: World Health Organisation, 1976.

23. Revankar CR, Dewarker PR, Singh Mulchand, Ganapati R. Leprosy in pre school age. Leprosy Review 1979; 50: 293 – 296.

24. Keeler R, Deen RD.Leprosy in children aged 0 – 14 years: Report of an 11 year control programme. Leprosy Review 1985; 56: 239 – 248.

25. Norman G, Joseph GA, Udayasuriyan P, Samuel P & Venugopal M. Leprosy case detection using school children. Leprosy Review 2004; 75: 34 – 39.

26. Kabir Sardana. A Study of Leprosy in Children, From a Tertiary Pediatric Hospital in India. Leprosy Review 2006; 77: 160 – 162.

27. Renu Roy, Kalla G. Pattern of leprosy in Children in Jodhpur. Indian Journal of Leprosy 1997; 69(2): 199 – 200.

28. Virendra N Sehgal, Anup K Chaudhry. Leprosy in Children; A prospective study. International journal of Dermatology 1993; 32: 194 – 197.

29. Dave DS, Agarwal SK. Prevalence of Leprosy in Children of Leprosy Parents. Indian journal of Leprosy 1984; 56(3): 615 – 621.

30. Michel F Lechat, Etinenne E. Declercq, Control programs in leprosy. In Hastings RC,ed.Leprosy, second edition, Edinburgh: Churchil livingstone, 1994 : 370.

31. Rene RP de Vries & Tom HM Ottenhoff. Immunogenetics of Leprosy. In Hastings RC,ed. Leprosy, second edition, Edinburgh: Churchil livingstone, 1994 : 115.

32. Dharmendra. Classifications of Leprosy. In Hastings RC, ed. Leprosy, second edition, Edinburgh: Churchil livingstone, 1994 : 179 - 189.

33. Prasad PVS. Childhood Leprosy in a rural hospital. Indian journal of Paediatrics 1998; 65(5): 751 – 752.

34. Ganapathy R, Naik SS & Pandya SS. Leprosy among school children in greater Bombay: Clinical Features. Leprosy Review 1976; 47: 133 – 140.

35. Kumar Vijay, Baruah MC, Garg BR. Childhood leprosy – A clinico epidemiological study from Pondicherry. Indian Journal of Dermatology, Venreology and Leprology 1989; 55: 301 – 304.

36. Pailoor Jayalakshmi, Tong M, Santokh Sing, Ganesapillai T. Leprosy in children. International journal of leprosy and other mycobacterial diseases 1997; 65; 95 –97.

37. Wesley S Ramani, Nair Gopalakrishnan TV, Nair BKH. Leprosy among school children in Trivandrum city. Indian journal of Dermatology, Venereology and Leprology 1990; 56(4): 286 – 298.

38. Sandra L Cortes, Rodriguez G. Leprosy in children: Association between clinical and pathological aspects. Journal of tropical paediatrics 2004 ; 50(1): 12 – 15.

39. Dayal R, Paliwal AK, Prasad R, Mathur PP, Bharadwaj VP, Girdhar BK, Pandey PN. A clinico-bacteriological profile of leprosy in children. Indian Paediatrics 1989; 26: 126 – 128.

40. Virendra N Sehgal. Leprosy in children. In Clinical leprosy, Fourth edition, Jaypee Brothers, Medical publishers (P) Ltd. New Delhi 2004: 78 – 82.

41. Virendra N Sehgal, Sumil Sehgal. Leprosy in young urban children, International journal of Dermatology 1988; 27(2): 112 – 114.

42. Jain S, Reddy RG, Osmani SN, Lockwood DNJ, Suneetha S. Childhood 3 leprosy in an urban clinic, Hyderabad, India: clinical presentation and the role of household contacts. Leprosy review 2002; 73: 248 – 253.

43. Sehgal VN. Leprosy in children. Leprosy review 1990; 61(2): 194.

44. Tarun K Chaudhury, Sabyasachi Majumdar. Clinicopathological determinants of indeterminate leprosy. Indian journal of Dermatology 1996; 41(3): 93 –98.

45. Sehgal VN, Srivastava G. Indeterminate leprosy: A passing phase in the evolution of Leprosy. Leprosy review 1987; 58: 291 –299.

46. Roy E. Pfaltzgraff, Gopal Ramu. Clinical Leprosy. In Hastings RC,ed. Leprosy, second edition, Edinburgh: Churchill livingstone, 1994: 237 – 284.

47. Jopling WH, McDougall. The disease. Handbook of Leprosy, Fifth edition. CBS Publishers and Distributors 1996: 22 – 50.

48. Prasad PVS. Clinical manifestations. In All about leprosy, First edition, Jaypee Brothers, Medical publishers (P) Ltd, New Delhi 2005: 49 – 61.

49. Anthony Bryceson, Roy E. Pflatzgraff. Symptoms and Signs. Leprosy, Third edition, Churchill livingstone 1991: 25 – 55.

50. Ricardo Fakhouri, Mirian N Sotto, Marli IP Manini, Leontina C Margarido. Nodular Leprosy of childhood and Tuberculoid Leprosy: A comparative, morphologic, immunopathologic and quantitative study of skin tissue reaction. International journal of Leprosy and other Mycobacterial diseases 2003; 71(3): 218 – 225.

51. Dayal R, Gupta R, Mathur PP, Katoch VM, Katoch K, Dhir GG. Study of gene probes in childhood leprosy. Indian journal of paediatrics 1998; (65): 99 – 105.

52. Sehgal VN, Joginder. Leprosy in children: correlation of clinical, histopathological, bacteriological and immunological parameters. Leprosy review 1989; 60: 202 – 205.

53. Roy E. Pfaltzgraff, Gopal Ramu. Clinical Leprosy. In Hastings RC,ed. Leprosy, second edition, Edinburgh: Churchil livingstone, 1994: 263 – 265.

54. Sujai Suneetha, Arunthathi Sigamani, Nisha Kurian, Chinoy JG Chacko. The development of cutaneous lesions during follow up of patients with primary neuritic leprosy. International Journal of Dermatology 2005; 44: 224 – 229.

55. Purohit S, Kalla G, Roy R, Batra A. Histoid leprosy in an eight year-old child. Indian Journal of Leprosy 1997; 69(4): 399 – 400.

56. Virendra N. Sehgal, Ashok Aggarwal, Govind Srivastava, Neelima Sharma, Sonal Sharma. Evolution of histoid leprosy (de novo) in lepromatous (multibacillary) leprosy. International Journal of Dermatology 2005; 44: 576 – 578.

57. Swapan K Samata, Nilajen Bhowmic, Debashih Patua, Sangeta Maity. Ocular leprosy in children. Indian journal of leprosy 2005; 76: 60 –61.

58. Bikash Ranjan Kar, Gigi Ebenezer, Job CK. Penile tuberculoid leprosy in a five-year-old boy. Indian journal of Dermatology, Venereology and Leprology 2005; 71: 125 – 127.

59. Mahajan PM, Jogaika DG, Metha JM. Study of deformities in children with leprosy – An urban experience. Indian journal of leprosy 1995; 67: 405 – 409.

60. David E Elder, Walter F Lever. Lever's Histopathology of skin. Eighth edition Lippincott, Williams & Wilkims, 1997: 477 – 486.

61. Charles K Job. Pathology of Leprosy. In Hastings RC,ed. Leprosy, second edition, Edinburgh: Churchill livingstone, 1994: 199 – 206.

62. Job CK, Sushil M Chandy. Differential diagnosis of leprosy – A guide book for histopathologist. Published by Karigiri Leprosy Education Programme, Schieffelin leprosy research & training center, Karigiri, Tamilnadu, India 2001: 32 – 64.

63. David Weedon. Skin pathology, second edition, Churchill livingstone, 2002: 602 - 603.

64. Charles K Job. Pathology of Leprosy. In Hastings RC,ed. Leprosy, second edition, Edinburgh: Churchill livingstone, 1994: 207 – 210.

65. Job CK, Sushil M Chandy. Differential diagnosis of leprosy – A guide book for histopathologist. Published by Karigiri Leprosy Education Programme, Schieffelin leprosy research & training center, Karigiri, Tamilnadu, India 2001: 77 – 88.

PROFORMA

Name: Date:

Age: Registration Number:

Sex: Hospital Number:

Informant:

Studying/Working:

Address:

COMPLAINTS:

Duration:

HISTORY OF PRESENT ILLNESS:

Skin Lesions

Anesthesia

Pain over the region of nerve

Impaired sensation over hands and feet

Inability in using hands and feet

Ulcers and blisters

Fever

Edema hands and feet

Joint pain

Stuffiness of nose

Epistaxis

Pain in and around eyes

Redness of eye

Photophobia

PAST HISTORY:

FAMILY HISTORY OF LEPROSY:

TREATMENT HISTORY:

IMMUNISATION HISTORY:

GENERAL EXMINATION:

Built:	Weight
Anemia	BCG Scar
Clubbing	Cyanosis
Edema Hands and Feet	Jaundice
Gynaecomastia	Lymphadenopathy

SYSTEMIC EXAMINATION:

Cardiovascular System:

Respiratory System:

Central Nervous System:

Abdomen:

DERMATOLOGICAL EXAMINATION:

Skin Lesions:

Number:

Size:

Site:

Morphology:

Colour:

Sensation:

Margin:

Surface:

Consistency:

Satellites:

Central healing:

Loss of hair:

Loss of sweat:

Scales:

Ulceration:

Local cutaneous nerves:

Tenderness:

Nails Hair

Palms and Soles Mucosa

PHERIPERAL NERVES:

Truncal Nerves:

Cutaneous Nerves:

VOLUNTARY MUSCLE TESTING:

SENSORY TESTING:

DEFORMITIES:

CLINICAL DIAGNOSIS:

INVESTIGATION:

Hemoglobin	Total Count
Differential count	Erythrocyte Sedimentation Rate
Total Protein	

SKIN SMEAR:

SKIN BIOPSY:

NERVE BIOPSY: